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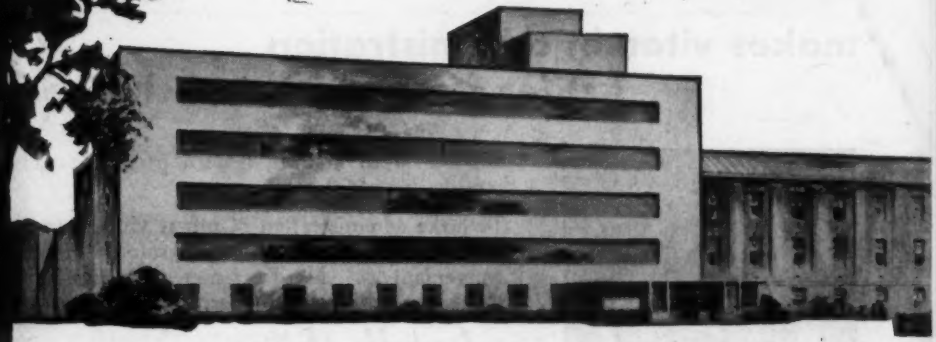

VOLUME XI *January 1955* NUMBER 1

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Clinical Proceedings

OF THE CHILDREN'S HOSPITAL

WASHINGTON, D. C.

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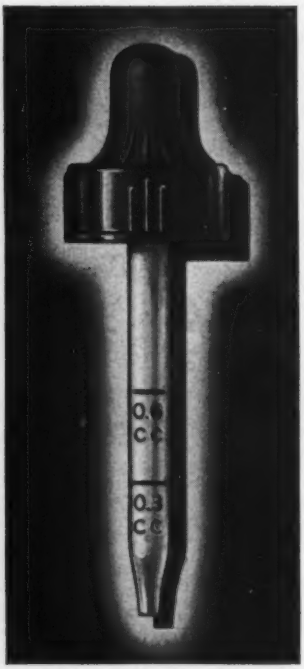

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PUBLISHED MONTHLY BY THE STAFF AND RESEARCH FOUNDATION OF THE CHILDREN'S HOSPITAL, WASHINGTON, D. C.

Cases are selected from the weekly conferences held each Sunday morning at 11:00 A.M., from the Clinicopathological conferences and from the monthly Staff meeting.

This bulletin is printed for the benefit of the present and former members of the Attending and Resident Staffs, and the clinical clerks of Georgetown and George Washington Universities.

Subscription rate is $2.00 per year. Those interested make checks payable to "Clinical Proceedings Dept.," The Children's Hospital, Washington, D. C. Please notify on change of address.

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Entered as second class matter November 21, 1946 at the post office at Washington, D. C., under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 538, Act of February 28, 1925, authorized January 17, 1947.

SHOULD A CHILD WHO HAS A HISTORY OF CONVULSIONS BE IMMUNIZED AGAINST PERTUSSIS?

Joseph Seebode*

Pertussis is still a challenge to the medical profession. It is a very serious disease when it occurs in infants under one year of age, or when complicated by pulmonary or cerebral lesions. Over 200,000 cases of whooping cough are reported annually in the United States with 5,000 to 7,000 deaths. The mortality rate is highest in those less than a year of age.

CEREBRAL COMPLICATIONS

The incidence of cerebral complications in pertussis is difficult to establish; however, Litvak[18] in a series of 6,000 cases of pertussis found 47 to have cerebral complications, an incidence of 0.8 per cent. Though the general mortality rate dropped from 9.4 per cent in 1938 to about 3 per cent in 1946, the mortality rate in patients with cerebral complications has not been materially changed. When convulsions complicate the disease the mortality rate is about 60 per cent. The incidence of cerebral complications in pertussis is higher in females than in males. Fifty per cent of patients who develop cerebral complications are under one year of age.

Theories regarding the pathogenesis of cerebral complications are numerous. They include as causes cerebral hemorrhage, angiospasm, anoxemia, edema and congestion of the brain, virus encephalitis activated by the disease, air embolism due to ruptured alveoli, tetany, and neurotoxicity.

An outline of the clinical patterns of cerebral complications in pertussis follows:

1. Convulsion and coma:
   a. continuous or recurrent
   b. generalized or local
   c. death during the attack
   d. survival with stupor
   e. coma persisting for days or weeks.
2. Paralysis
   a. cerebral type
      1. *motor:* monoplegia, diplegia, hemiplegia, paraplegia
      2. *sensory:* aphasia, deafness and blindness.
   b. spinal type
      1. spastic
      2. flaccid
3. Meningeal syndrome

* Class of 1955, Georgetown U. School of Medicine.

4. Neurological signs
5. Peripheral neuritis
6. Mental changes
7. Epilepsy

The prognosis is very limited. The mortality rate when cerebral symptoms are present is about 40.4 per cent. In general it may be stated that one-third recover to develop sequelae and one-third recover completely. Sequelae include mental retardation, paralysis, recurrent convulsions, loss of visual acuity, tone deafness, speech disorders, ataxia, personality and behavior changes, delay in walking and talking, epilepsy, idiocy, hemiplegia, or spastic paraplegia, hydrocephalus and diffuse cerebral atrophy.

All of the above facts are mentioned in order to emphasize the importance of prevention of this disease [18].

EFFICACY OF VACCINE

In order to come to a conclusion regarding the use of pertussis vaccine in convulsive children we must also have some idea of the effectiveness of the vaccine in preventing pertussis. One of the first large controlled studies on the subject concerns outbreaks on the Faroe islands. Epidemics on the islands in 1923 and 1929 prompted the artificial immunization of 3926 people. Of the vaccinated group, six died, a mortality of 0.15 per cent. In a non-vaccinated group of 1,073 there were 26 deaths, a mortality of 2.4 per cent. It was also noticed that the disease in vaccinated individuals was much milder and of shorter duration. It was concluded that prophylactic immunization against pertussis should continue but it was not recommended for children under one month of age.

A recent study on the prevention of whooping cough by vaccination was done by the Medical Research Council Investigation in 1951 in Great Britain. Groups of children between six and 18 months of age were randomly selected. Two groups of equal size were vaccinated. One was vaccinated with pertussis vaccine and the other with an anti-catarrhal vaccine. The former group included 3,800 individuals while the other group contained 3,750. None developed convulsions following immunization. Of the vaccinated group 149 developed clinical pertussis and of the control group 687 developed clinical pertussis. This represents a reduction in the incidence of disease of 78 per cent. There was a clinical attack rate of 18.2 per cent among children who were immunized and 87.3 per cent in the controls. During two or three years following immunization there was no reduction in the degree of protection.

VACCINE ENCEPHALOPATHY

From what has been said thus far concerning the disease and its prevention there would seem to be no doubt as to the general advisability of

pertussis immunization. However, it is a well established fact that encephalopathies may follow the injection of various serums and vaccines. Isolated instances of reactions following the prophylactic use of pertussis vaccine have been reported since 1933 when Madsen[1] described two deaths which occurred in Denmark following pertussis vaccination. In both cases, the children were new-borns in families in which there were other cases of whooping cough. In the first case an injection was given on the first day of life and a second four days later. One half hour after the last injection, contraction in the arms and legs occurred; cyanosis, hiccough, convulsions and death followed within a few minutes. In the second case the first injection was given eight days after birth and the second three days later. Two hours after the last injection the child died suddenly with slight cyanosis but no convulsions or vomiting. At postmortem examination no definite cause of death could be established[1].

In 1936 Doull, Shibley, and McClelland[2] reported three untoward reactions in a series of 500 children receiving pertussis vaccine. These three children are said to have had convulsions within twelve hours following inoculation. Small elevations in rectal temperature were noted in about fifteen of the cases. Taylor[3] described the case of a ten month old normal boy who received one cc. of Sauer[15] pertussis vaccine in each arm in the upper deltoid region. Five hours after the injection the child became irritable and cold and developed tonic contractions of both feet. Both lower extremities were limp. The contractions lasted ten minutes. No medications were given and the child recovered completely. Since this reaction followed the first injection it was not thought to be caused by sensitization but rather by the direct action of the vaccine on the central nervous system[3].

Kendrick and Eldering[4] reported the immunization of 1,815 children, six to fifteen months of age with only one reaction characterized by convulsions. Sako and his co-workers[5] reported a series of 6,600 children who received injections of H. pertussis alum precipitated vaccine which included two cases associated with vomiting, diarrhea and convulsions.

Werne and Garrow[6] in 1946 reported the death of identical twins from anaphylactic shock following the second injection of diptheria toxoid and pertussis antigen, alum precipitated. They were both boys aged ten months. There was no previous history of convulsions. Their first injection, given one month before, was followed by a low grade fever and slight vomiting. Following the second injection there was irritability and vomiting within an hour and both died in shock within twelve hours. There were no convulsions seen and histopathologic study at autopsy revealed lesions consistent with anaphylactic shock namely arterial spasm and increased endothelial permeability. Sensitization seems to have played a part in this reaction, rather than any specific toxin[6].

In 1947 Brody and Sarly [7] reported another death following the last

injection of pertussis vaccine. The first two injections were accompanied by somnolence, constipation and difficulty in urination. The third injection was followed by death within twelve hours. Sensitization also seems the most likely pathogenesis in this case. Because of the scarcity of mesenchymal tissue in the central nervous system it seems likely that the locus is in the neurovascular system. These authors conceded that all children should be immunized but considered the appearance of neurological symptoms within two weeks a contraindication to further injections.

One of the most thought provoking reports on this subject and one which introduces the dilemma with which we are dealing was that of Byers and Moll [8] in 1948. Inspecting the records of the Boston Children's Hospital over a ten year period they discovered fifteen instances in which children developed acute cerebral symptoms within a period of hours after the administration of pertussis vaccine. Twelve of the children were boys and three were girls. At the time of injection they ranged in age between five and eighteen months. All had a history of normal development. There had been no convulsions before injection. In three cases however, there was a family history of convulsions. Many different lots of vaccine were implicated. The only common precipitating agent was some form of pertussis vaccine. Fluid pertussis vaccine was used in twelve instances, combined diptheria toxoid in two cases and triple antigen in one case. The usual dosage schedule of 20 billion, 40 billion and 40 billion bacteria, or less was used in all instances save one in which an initial dose of 40 billion bacteria was followed in a matter of minutes by irritability, coma, fever and convulsive reaction. In addition three children who had convulsive reactions to an earlier dose, reacted to a succeeding dose with convulsions.

In this series the reactions developed as soon as twenty minutes and as long as seventy-two hours after the injections. The outstanding symptom was convulsions. Changes in consciousness varying from drowsiness or irritability to deep coma occurred in all. On admission to the hospital all had neurological signs; five were described as comatose; four had hemiplegia; and two had generalized hyperreflexia with bilateral clonus and Babinski's signs. The acute illness varied from thirty-six hours to ten days. Two years later one child who had three convulsions over a five month period had remained well and development seemed normal. The remaining fourteen children suffered from a variety of sequelae. One child had not regained his speech ability which was present before injection. Another infant had a remission of convulsions for four months following pneumoencephalography. Another patient developed normally for four months only to enter status epilepticus at the onset of a respiratory infection when fourteen months old. From this time he pursued a retrogressive course ending in death in an institution. The remaining children showed varying

degrees of marked retardation. On psychological examinations there were marked defects of attention, irregularities of mental development most marked in relation to abstract behavior and difficulty in organizing material. Three were grossly defective, of whom one died in an institution. Recurrent convulsions continued over variable lengths of time in thirteen children.

Of eight pneumoencephalograms done during the follow-up period, six showed dilatation of the ventricular system and two were at the upper limit of normal. Of ten electroencephalograms, six were normal and four abnormal.

During the same period in which the above study was done twenty-six children were admitted to the Boston Children's Hospital suffering from acute or chronic effects of encephalopathy accompanying pertussis itself. Half of these children were seven months old or younger and had not been immunized because it was just not the practice at that time. Of the older group only one had a course of vaccine ending approximately six weeks prior to onset of her pertussis. Of the twenty-six children, six developed normally, seven died of their acute illness, three could not be found and the remaining ten were sufficiently damaged to make competitive living unlikely.

In conclusion the authors stated that, "it seemed likely that babies are safer vaccinated than not; however, universal use is warranted only if it can be shown to be effective in preventing encephalopathy or death from pertussis. If avoidance of the inconvenience of the average attack of pertussis is all that is expected the risk seems considerable."

Toomey [9] in 1949 reported a series of thirty-eight well authenticated cases of reactions to pertussis vaccine. Two had severe reactions and died; four had subsequent convulsions; four were simultaneously ill with other diseases and five had convulsions previous to injection. Twelve showed irreversible changes. No common denominator was found, fluid toxoid having been given with the greatest frequency. Of all patients seen in Cleveland and vicinity none had irreversible changes.

Possible causes of these reactions were listed as follows: (1) Constitutional reactions related to host factors; (2) Immediate intravenous injection of vaccine; (3) Antigen-antibody responses to previous specific sensitization which is not likely since reaction often follows first injection; (4) Process of pyrogens in bacterial organisms; (5) Damage of cerebral vessels from toxins in pertussis organisms.

In summary they stated: (1) No child should receive injections of vaccine in large amounts (40 billion organisms) who had (a) a family history of convulsions, (b) present history of convulsions, (c) illness of any kind but particularly in central nervous system; (2) If there is any doubt the

dosage should be decreased; (3) A report should be made of every apparent reaction and date should be kept by the physician; (4) Perhaps the U.S. P.H.S. should standardize vaccine not only for potency but for aging*; (5) There are not enough reactions to discontinue the use of the vaccine.

Sauer commented in the above article that since pertussis is often the initial immunization procedure for a child it has become a factor which precipitates fever, convulsions and encephalopathy in infants with potentially unstable central nervous systems. The trigger effect of any fever producing antigen may cause this typical chain of symptoms. Hoyne of Chicago suggested the possibility that untoward results were due to the activation by the pertussis vaccine of some virus harbored by the patient.

Anderson [10] in London described a case of encephalopathy in a two and one half year old boy. The reaction occurred thirty-six hours after the first injection. There was a history of convulsions. He concluded that vaccination with pertussis is contraindicated where there is a family history of convulsions or any untoward reaction following the first injection.

Grace [11] from London, Ontario, discussed two cases in which encephalopathy followed injection of pertussis vaccine. In both cases combined antigens were used including fluid forms of diphtheria toxoid, pertussis vaccine and tetanus toxoid. The pertussis component was considered responsible. In one case an explosive episode occurred with severe, prolonged convulsive seizures and right sided hemiplegia nine hours after the first dose of antigen. Sequelae remained as an indication of permanent cerebral damage. In this case there was a family history of convulsions. In the second case there was a past history of convulsions with an attack of tonsillitis and also a family history of convulsion. This article again draws attention to the serious results which may occur when pertussis antigen is administered to a subject who is predisposed to the convulsive state.

In follow-up studies on pertussis in infancy made by Byers and Pizza[12] in 1950 the following conclusions were reached: "since pertussis vaccine seems prophylactically efficacious, the risks of its use in infancy are probably outweighed by those of the disease itself."

Miller[13] reviewed the literature on this subject in 1951 and came to the following conclusions: (1) pertussis vaccine is contraindicated in any acute illness; (2) it should not be given to children with cerebral damage; (3) children with a convulsive history should be given fractional doses. He also was of the opinion that the alum precipitated and aluminum hydroxide adsorbed vaccines were preferable. He recommended that protein in one injection should be limited to a cell count of twenty-billion organisms.

* See editorial this issue.

At about the same time in Colindale, England, Cockburn[14] studied the problem of pertussis immunization. He was aware that 9,401 deaths had occurred in England and Wales in the past ten years. After reviewing the American and British literature the following provisional recommendations were proposed: (1) vaccination should not be employed on a country-wide basis until potency of vaccine can be reliably assessed by means of a laboratory test; (2) plain suspension of killed bacteria should be employed in preference to alum precipitated or combined vaccines; (3) vaccination should not be carried out when the incidence of poliomyelitis is high in an area; (4) children with a personal or family history of organic nervous disease should not be inoculated; (5) children should not be inoculated if they are known to be convalescent from any of the common fevers particularly those due to viral infections; (6) children should not be inoculated during the incubation period of any common fevers if they are known to have been exposed and if they are thought to be susceptible; (7) vaccination should not be attempted before the age of six months and should be carried out after diphtheria immunization which is the more important; (8) if the reaction to the first inoculation is severe, or if the child develops convulsions at any time during the inoculation procedure, no further inoculations should be given; (9) the dosage should be that recommended by the manufacturer of the vaccine. In general, three doses, each of 0.5 ml., or 1 ml., depending upon the concentration of bacteria in the vaccine are given at monthly intervals.

In 1953 Sutherland[15] discussed toxic reactions to pertussis vaccine and theories of etiology. He proposed an allergic or pathergic state of the central nervous system as a possible precursor. He also felt it was possible that latent neurotropic viruses may be activated. His conclusion was that the benefits derived from immunization outweighed the risk involved but risks may be minimized by careful assessment of history and clinical condition of the child.

Sauer[17] in 1953 reviewed the literature and made the following recommendations concerning immunization: (1) the slowly absorbed alum containing "DTP" is preferred to the fluid toxoid because it produces earlier, higher and longer sustained immunity responses than does pertussis vaccine in isotonic sodium chloride; because of delayed absorption, febrile reactions are less frequent and milder. In postencephalitic encephalopathy, the vaccine in isotonic saline was more frequently implicated than D.T.P.; (2) any child with a history of convulsions should not be given the injection. Should immunization seem desirable however, fractional doses of 0.2–0.3 cc. may be tried at monthly intervals until full dosage has been accomplished; (3) D.T.P. immunization should be deferred in premature

and frail infants and those with fever or pustular infections of the skin or ears.

In December of 1953 a new light was thrown on the problem when Melin reported on the immunization of children at the Clinic for Convulsive Diseases in Stockholm, Sweden. He had a completely different approach to the problem. Two cases were cited in which patients with convulsive disorders developed pertussis and had exacerbation of their convulsive disease. Ten such cases have been collected. One case was in identical twins who had almost identical petit mal seizures. Both were well controlled with tridione. One developed pertussis as a result of which his seizures have always been worse than before the disease in spite of the many drugs which have been tried. Of 48 children with different convulsive disorders and 30 mongoloids only one developed complications of pertussis immunizations. After the second injection she had major convulsions on three successive days and two years later presented delayed speech development. In conclusion he recommends that pertussis or triple vaccination be given to children with convulsive disorders. He does recommend that immunization be interrupted at the first signs of any complication. If high fever follows an injection, phenobarbital could be given or pre-existent anticonvulsive medication could temporarily be increased[16].

Several physicians were asked to comment on Melin's contention. Samuel Livingston, chief of the Epilepsy Clinic at Johns Hopkins Hospital agreed with Melin's recommendations. In his series of 7,000 epileptic children 11 developed seizures following routine prophylactic immunizations. Seven of these began having recurrent seizures soon after the administration of pertussis immunizations. None of these 11 had suffered with convulsions and there was no family history of epilepsy. In a series of 686 patients with idiopathic epilepsy who were given pertussis vaccine there was not one with significant reactions. Ninety-six patients who had suffered with simple febrile convulsions were given pertussis inoculations. Ten had a recurrence of simple febrile convulsions soon after the injection but over a ten year period none developed epilepsy. Two hundred eighty-four patients with secondary epilepsy were immunized and in no case were any apparent permanent effects encountered. In conclusion he did not feel that one can predict which child is more likely to be adversely affected by pertussis inoculations. "There is a much greater chance of increasing the epileptic population by allowing a child to contract pertussis than by the administration of pertussis inoculations."

William Lennox of The Neurological Institute of the Children's Medical Center in Boston is not nearly so convinced as Melin or Livingston. He feels that convulsions are not so much to be feared as cerebral pathologic lesions, and that mental retardation is as serious a complication as con-

vulsions. Hence a known existing lesion or an allergy to immunizing agents should carry a greater warning than lowered threshold to convulsions. Lennox seems particularly to remember convulsions following prophylactic injection. This may be because complications, that are induced by the physician rather than nature remain more vividly in the memory. He feels that more study is necessary as to incidence of pertussis and complications in vaccinated as compared with unvaccinated individuals.

Byers agreed with Melin. He explains to the mothers of his children the problem and in almost all instances they have wanted to have their children vaccinated. He does hesitate when the child is defective and not active enough to be with other children to have a chance of contracting the disease[16].

The three theories which are most plausible in explaining the above reactions are: (1) the presence of a neurotoxin in the vaccine; (2) the activation of latent neurotropic viruses; (3) a reaction involving an antigen-antibody response. The last seems unlikely because many reactions have followed the first injection, making it difficult to explain how the host could have become sensitized. Very little work has been done concerning the activation of neurotropic viruses and it is impossible to come to any conclusion at this time. As for the toxin theory considerable work has been done. Fontagne and Dagnelie[8] prepared an endotoxin which produced a diffuse encephalitis and death when injected into either the cerebral substance or subarachnoid space of rabbits or guinea pigs. The heated pertussis endotoxin used in the same way did not reproduce the results. Nahamara produced encephalitic changes in rabbits with intracutaneous injections of cultures of *H. pertussis*. He also produced both an endotoxin and an exotoxin from the same cultures which when injected into the subarachnoid spaces of rabbits, caused a rapidly fatal encephalitis. It would seem that more work needs to be done in evaluating these toxins and also in modification of the vaccine or the study of new methods of administration.

SUMMARY

Data has been presented on the use of pertussis vaccine over the past twenty years. Over 200,000 cases of whooping cough are reported annually in the United States with 5,000 to 7,000 deaths. It is still the most important of all infectious diseases from the point of view of morbidity and mortality. Of all children who develop pertussis, 0.8 per cent develop cerebral complications and of this group about 60 per cent do not recover. The vaccine may reduce the incidence of clinical pertussis 78 per cent. The dilemma with which we are dealing results from the fact that many deaths and irreversible cerebral complications have been reported following inoculation with pertussis vaccine. There are reports from neurologists, pediatri-

tricians, immunologists and general practitioners. The neurologists who have inoculated large series of epileptic children and those with convulsive disorders are unable to see any contraindications. The pediatricians are hesitant because they have inoculated more children and have reported more reactions. The general practitioners seem more impressed by reactions to the vaccine and find definite contraindications. Since a large number of children who have had reactions have a personal or family history of convulsions it is felt that such a history is a definite contraindication to use of vaccine.

CONCLUSION

This situation places the practitioner on the horns of a dilemma. If he decides to immunize the child with a history of a convulsive disorder and the child develops encephalopathy he will be blamed. If he does not immunize and the child develops pertussis complicated by encephalopathy he will also be blamed. The fact that more children are being immunized all the time does reduce the risk of a non-vaccinated child developing pertussis.* For one decision he is backed by experienced workers in the field of neurology and for the other he is backed by immunologists and pediatricians. It does not seem with our present state of knowlege that malpractice can be charged if there is an unfortunate ending in either direction. It comes down to an individual problem requiring that the situation be explained to the parents. It appears that certain precautions should be taken and in doubtful cases, immunizations when definitely indicated should be started cautiously with fractional doses and immunization stopped at the first sign of central nervous system symptoms.

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ACUTE GLOMERULONEPHRITIS WITH CONGESTIVE HEART FAILURE

CASE REPORT

James Stallings, M.D.
Frederic Burke, M.D.

A. S., a ten year old white girl, was admitted to Children's Hospital at 10 a.m. on December 18, 1953, following a generalized convulsion. She had been well until two weeks before admission when there developed a cough, runny nose, and sore throat associated with low grade fever. These symptoms gradually subsided and she was relatively well until three days before admission when she began to pass dark, smoky urine, developed puffiness about the eyes, and complained of cramping abdominal pain. She was examined by her local physican whose impression was that she had infectious hepatitis. On the evening before admission she complained of increasingly severe headache and later seemed "out of her head." At 6 a.m. on the day of admission she had a generalized convulsion which lasted about five minutes. Afterwards she began to vomit at frequent intervals. Past history and family history were non-contributory.

At physical examination she was acutely ill, disoriented, and retching almost continuously. She understood commands. Temperature was 99.8, pulse 120, respirations 20, blood pressure 166/110, and weight 80 pounds. No periorbital or pretibial edema was noted. Tache cerebrale was present. No abnormalities were noted on funduscopic examination. Mild pharyngitis was noted, but the remainder of the examination was normal.

The urine was grossly bloody, with specific gravity of 1.002 and 240 mg. per cent albumin; microscopic examination showed a few granular casts and many red and white blood cells. Hemoglobin was 11.5 gm. per cent; hematocrit 38 per cent; WBC

19,200 per cu. mm. with 85 per cent segmented forms, 7 per cent lymphocytes, 8 per cent monocytes. Blood urea nitrogen was 17 mg. per cent and sedimentation rate 33 mm. per hour (Wintrobe). Lumbar puncture yielded clear fluid under pressure of 214 mm. of water; the protein was 22 mg. per cent, glucose 85 mg. per cent, and there was less than one cell per cu. mm.

After 60 mg. phenobarbital and 8 cc. of 50 per cent magnesium sulfate intramuscularly the patient became more rational and the blood pressure fell to 140/100. Ten hours after admission she became dyspneic; cardiomegaly was noted on percussion. An electrocardiogram at this time was within normal limits. Chest x-ray confirmed the presence of cardiomegaly and pulmonary congestion (fig. 1). She was treated with 0.8 mg. digitoxin intramuscularly in divided doses. The elevated blood pressure responded to doses of 5 cc. per kilogram of 3 per cent magnesium sulfate solution intravenously repeated every six to eight hours as necessary. These medications

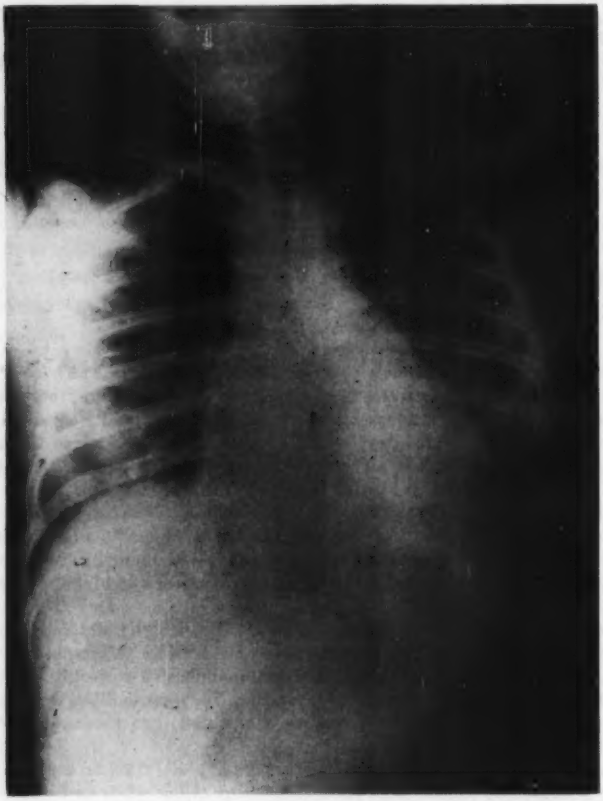

FIG. 1. Cardiomegaly and pulmonary congestion in a patient with glomerulonephritis.

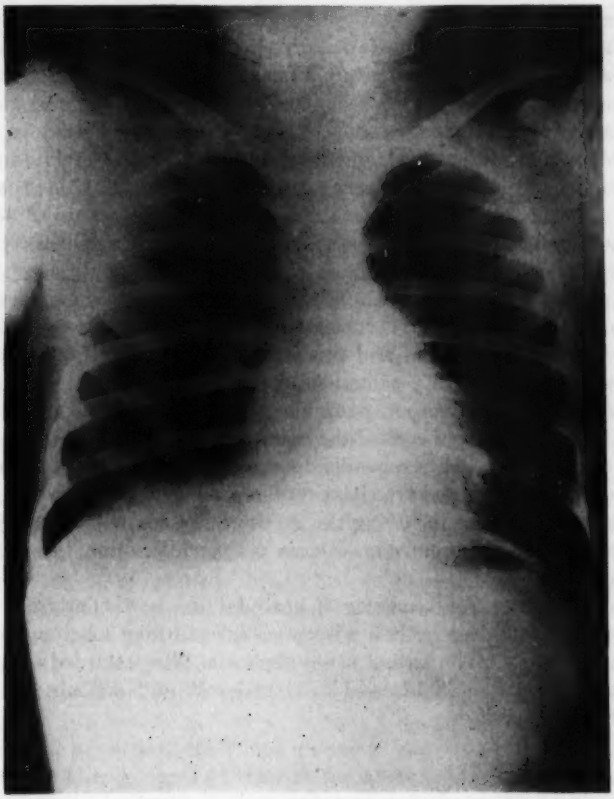

FIG. 2. Resolution of cardiomegaly and pulmonary congestion following the use of digitalis and magnesium sulfate.

were discontinued on December 21, the third hospital day, and recovery thereafter was uneventful. Repeat chest x-ray on December 22, (fig. 2) was within normal limits.

COMMENTS

Frederic Burke, M.D.

This case illustrates most of the classic features of glomerulonephritis as this disease is seen in children. Edema, hypertensive convulsions, abdominal pain and gross hematuria, following an upper respiratory episode by a few days, presents the most typical picture that is observed. Edema which may be minute or absent in about 20 per cent is the most common presenting symptom and is detectable in 80 per cent. The second most com-

mon presenting symptom is gross hematuria, and the third is abdominal pain. Acute cerebral manifestations such as convulsions, frontal headaches, and coma are present in about 20 per cent of the cases, and are usually accompanied by one or more of the other striking and alarming symptoms of this disease.

Evidence of the heart involvement with nephritis is to be found in direct proportion to the level of awareness of the examiners that this complication may occur. Indeed, heart failure is the commonest cause of death in the 5 to 6 per cent of children who succumb to acute nephritis. Cardiac dilatation which returns to normal size while in the acute convalescent phase appears in 70 per cent. Congestive heart failure characterized by dilatation and pulmonary congestion demonstrated by x-ray, elevated venous pressure, dilatation and pulsation of the cervical veins, enlargement of the liver (with production of abdominal pain), and abnormal electrocardiographic changes, particularly inversion of T waves, will be present in at least 50 per cent of the cases.

The cause of heart failure in this disease has not been fully defined but the concept of generalized arteriolar capillary damage of the kidney, brain, liver and other organs including the myocardium seems most acceptable. Hypertension and hydremia may have a trigger function to exceed the cardiac reserve.

Consideration of the frequency of heart failure, particularly in relation to mortality, would make it a wise procedure to take maximal steps to protect the heart in all cases of acute nephritis. Where needed evidence is present, limitation of fluids and salt, bed rest and digitalis should be promptly instituted.

DIAGNOSTIC BRIEFS

COARCTATION OF THE AORTA

John O. Nestor, M.D.

A *coarctation of the aorta* is a constriction at any point between the arch and the bifurcation although about 98 per cent of them occur just below the origin of the left subclavian artery. Moreover, there may be considerable variation in the length and degree of narrowing of the coarcted area. In the so-called infantile type the coarctation is proximal to the entrance of the ductus arteriosus, and death usually occurs shortly after birth be-

cause a collateral circulation has not developed. The patient with adult or post-ductal coarctation usually survives because an adequate collateral circulation has developed. This is the common type under discussion.

The coarctation results in an elevation of the *blood pressure* in the upper part of the body in the vessels proximal to the coarcted area. Conversely the blood pressure and amplitude of pulsations are diminished in the lower part of the body. The nature of the hypertension is not entirely clear. It does not appear to be entirely due to mechanical obstruction and renal ischemia. In dogs it takes at least a 50 per cent constriction of the lumen of the aorta to produce significant changes in blood pressure and experience suggests that this probably applies in humans also.

An extensive *collateral circulation* develops between the branches of the subclavian and intercostal arteries and between the internal mammary and deep epigastric arteries. Inasmuch as the principal pathways of collateral circulation are by way of the posterior intercostal arteries, these become enormously enlarged and tortuous and press against the lower margin of the ribs where the pulsations over a period of years erode the ribs and produce notching detectable on roentgenogram.

There are frequently no *symptoms*; and incapacitating ones are not common early in life although these may develop at any age. When present they may consist of epistaxis, headaches, coldness, numbness or weakness of the legs, and eventually the complication of cardiac failure, cerebral vascular accident, or sub-acute bacterial endocarditis.

The *signs* result primarily from the disparity in blood pressures, the collateral circulation, and associated congenital cardiac lesions if the latter are present.

Normally, of course, the blood pressure taken by the standard cuff method is higher in the legs than in the arms but with a coarctation of the aorta this relationship is reversed, and there is a disparity in arterial pulsations between the upper and lower extremities. The femoral, popliteal, posterior tibial, and dorsalis pedis pulsations are weak and delayed or not palpable in contrast with the bounding pulse in the upper extremities. A difference of blood pressure and pulse between the two arms suggests that the left subclavian artery is involved in the area of coarctation. The collateral circulation may give rise to visible and palpable pulsations especially in the back between the scapulae.

The heart may or may not be enlarged depending on the degree of coarctation and the height of the blood pressure. Murmurs may be present and the common one is a harsh systolic murmur maximum in intensity along the left sternal border and in the left interscapular space. Occasionally there is a diastolic murmur resulting from aortic insufficiency which suggests the presence of a bicuspid aortic valve. The second aortic sound is

accentuated. A systolic thrill and a harsh systolic murmur maximum in intensity in the aortic area and radiating into the neck usually indicates the presence of a coexisting sub-aortic stenosis.

The findings on *roentgenographic examination* depend on the age of the patient and the degree of coarctation and hypertension. In infants and younger children the heart may appear normal but usually some degree of left ventricular hypertrophy can be demonstrated in the left anterior oblique view. The ascending aorta may be dilated and the aortic knob small or normal. Rib notching is not common until the second decade of life.

The *electrocardiogram* also may be normal in children but usually exhibits some degree of left ventricular preponderance or hypertrophy. *Special diagnostic studies* are usually not necessary to establish a diagnosis except occasionally in infants. An angiocardiogram or retrograde aortogram will demonstrate the location and extent of the lesion thus enabling the surgeon to decide if a graft is likely to be necessary and to estimate the length and size of the graft needed. Cardiac catheterization is of no value unless there are unusual coexisting congenital cardiac lesions.

Associated abnormalities which may complicate the picture are bicuspid aortic valve, aortic or sub-aortic stenosis, patent ductus arteriosus, septal defects, congenital mitral stenosis, and congenital aneurysms of the vessels at the base of the brain.

Before surgery became available to these patients, the average age at death was 35 years and nearly two-thirds were dead by the age of 40 years. Three-fourths of these die of the complications of the coarctation, namely, rupture of the aorta, sub-acute bacterial endocarditis, cardiac failure or intracranial hemorrhage. About one-fourth die of incidental causes, causes not related directly to the coarctation such as pneumonia, uremia, tumors, trauma, and coronary artery disease.

Surgical correction is the treatment of choice except in early infancy where conservative medical treatment results in a much lower mortality rate although this attitude has been questioned recently. The ideal age for operation is between 6 and 12 years but may be performed at any age if necessary. After 12 years of age the operation becomes more difficult and hazardous with advancing age because of arteriosclerotic changes and the enormous collateral circulation that develops. The overall mortality in all age groups should be less than 10 per cent and in children at the ideal age it should be well under 5 per cent.

Post-operatively there is striking improvement. Symptoms disappear and the blood pressure gradually returns to normal over a period of a few weeks. The great majority of patients are completely cured, a few obtain partial relief, and an occasional case obtains no relief from surgery.

The surgical procedure of choice is excision of the coarctation and primary anastomosis. Arterial homografts or plastic grafts are frequently necessary in adults. Occasionally it is practical to anastomose the left carotid or the left subclavian to the descending aorta.

CLINICOPATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.
Assisted by: Roger Bergstrom, M.D.
By invitation: Robert Parrott, M.D.

PROTOCOL

A white female infant was first seen at the age of two weeks because of the presence of cyst-like masses on the knees and a similar mass on the lateral aspect of the chest wall. These were not noted by the pediatrician when the infant was examined after birth. During the following week masses were noted to appear on the anterior surface of the right tibia, the calf of the left leg, the proximal portion of the anterior surface of the right thigh, the dorsal surface of the right hand and the scalp. These masses were freely movable subcutaneous nodules, which were firm and averaged 5 to 8 mm. in diameter. They were apparently nontender. Twenty-four hours prior to admission to the hospital blood was noted in the stools.

Past history revealed that the infant was born at eight months gestation and weighed four pounds, four ounces at birth. She gained weight steadily.

The patient's mother had had three miscarriages, the first at three months and the next two at two months gestation. There were no other siblings.

The laboratory tests showed negative blood Kahn and Mazzini tests, a normal hemoglobin and red cell count, a leucocytosis averaging 25,000 cells per cu. mm. of blood with 48 per cent of the cells being neutrophils and 52 per cent lymphocytes. Benzidine tests on the stool were strongly positive. Urinalyses were essentially normal except for a green reduction of Benedict's solution on two occasions.

X-ray examinations of the long bones showed a marked osteo-chondritis of the heads of each femur, tibia, fibula, and humerus; these changes were progressive.

A biopsy of one of the nodules was performed.

At four weeks of age severe diarrhea developed and despite intensive therapy, the infant lost weight rapidly and died at the age of six weeks.

Discussion: Dr. Parrott

There are several points in this protocol on which we might dwell:

1. The time of onset of illness is certainly early neonatal and I suspect that the process should be classified as congenital.

2. The nature of the skin lesions is such that they are noted gradually to develop; they are subcutaneous, cyst-like, non-tender and less than a centimeter in diameter. Their color is not noted, so I presume that they are not pigmented. They are described as being present in numerous areas of the body with no notation as to whether there is any peculiar configuration or correlation with neural distribution.

3. There is melena, a finding which may indicate infiltration of intestinal mucosa, whether by lesions similar to those on the skin or not.

4. There is rapid weight gain from a small newborn size, a factor weighing against generalized infection or metabolic illness.

5. There are progressive bone lesions early in the course of illness, another potential sign of infiltrative disease. These findings are quite symmetrical.

6. The family history includes nothing to suggest directly that this is a familial illness; however, the frequency of miscarriages leads one to suspect some process in the parents detrimental to the fruition of pregnancy.

7. Notable in the laboratory work are negative serological tests for syphilis, a lack of anemia, a moderate leucocytosis with normal differential count and a suggestion of reducing substance in the urine.

8. The baby's general condition is apparently fair until the development at four weeks of age of severe diarrhea, unresponsive to therapy, which apparently is the vanguard of death. This suggests the possibility of adrenal insufficiency potentially a manifestation here of an adrenal infiltrative process.

Thus we have a baby with the occurrence, probably at birth, of non-inflammatory skin masses, with progressive evidence of symmetrical involvement of the bones, the gastrointestinal tract (or possibly liver as a cause of bleeding), and eventually perhaps the adrenal glands with the production of shock.

Descriptively the skin lesions in this case, cyst-like, subcutaneous, and non-tender, might originate in misdirected growth of a number of tissues: hemangioma, lymphangioma, lipoma, sebaceous cyst, nevus, neuroblastoma, neurofibroma, lymphoma and xanthoma. They may also represent granulomatous nodules. If they are inflammatory, it is necessary to think of them among the more chronically inflammatory processes. Which of these then is most compatible with the description and which can cause bone, gastrointestinal and possibly adrenal infiltration.

Hemangiomas may indeed become generalized, causing infiltration of mu-

cous membranes, muscle, viscera, bone and central nervous system, but they are not apt to involve bones symmetrically. The skin is usually red or purple in color and we have presumed that the lesions in the protocol are non-colored.

Lymphangiomas usually are solitary; they situate on the neck, mucous membranes or proximal extremities usually; they are not likely to infiltrate the bones.

Lipomas may become generalized over the skin but, to my knowledge, they are not known to involve the osseous or other systems.

Sebaceous cysts would fit the description of the skin lesions in the protocol; however generalized disease due to sebaceous cysts is unknown to me.

Nevuses of the pigmented type may become malignant; however bone involvement as described in the protocol is not known.

Neuroblastomas: these embryonal tumors of adrenal sympathetic neural origin metastasize commonly and rapidly to the liver, skull and long bones; skin lesions are not common and would be unlikely to be the presenting complaint as is true in the protocol.

Lymphomas are capable of causing areas of "destruction and production" in the metaphyses of long bones. However that is not the type of lesion noted in the protocol; nor is there the anemia that would accompany leukemia or the primary lymph node lesions of lymphosarcoma or Hodgkin's disease. Neonatal occurrence of these tumors is also quite rare.

Xanthomas would be expected to be yellow in color and would, if "essential", be accompanied by added signs of Hand-Schüller-Christian syndrome. The secondary xanthomas do not usually affect bones.

Granulomas such as occur in sarcoidosis or tuberculosis or brucellosis, should be accompanied by other physical or environmental signs of these infections. For example the skin lesions of sarcoidosis are usually small, pink and do not have a cystic feeling; there should also be some chest signs and the bone lesions are more apt to be cystic and to occur in the hands. This would be almost too early in life to expect tuberculous infection to have a strong, fatal hold. Among the other infections which might have caused this child's death, low grade systemic fungus disease might be mentioned and congenital syphilis considered very seriously. The skin lesions of coccidioidomycosis or other systemic fungus infections are usually raised, red and tender. The bones are not often attacked but the lungs are frequently a site of disease. Skin tests, cultures and serologic procedures might have been employed to eliminate one of these unlikely etiological agents.

If we assume for a moment that the Kahn and Mazzini tests are falsely negative, a possibility but not a probability, how would this child's picture simulate congenital syphilis? There is a suggestive history of multiple mis-

carriages. The greatest suggestion is in the x-rays. From what we can see, there is symmetrical and universal involvement of all the long bones visualized. There even appears to be a typical Wimberger's sign, symmetrical destructive metaphyseal lesions on the medial aspects of the tibias.

However, the skin lesions described are not the typical maculo-pustular rash of syphilis which often affects the palms and soles. Nasal discharge, dry skin, rhagades, hepato-splenomegaly or anemia are not described. The absence of these signs and the negative serological test for syphilis, therefore, deter me from considering congenital syphilis as a likely diagnosis.

You may have noted that in discussing skin lesions which also cause systemic disease I have until now omitted neurofibroma. This tumor whose cell origin is apparently neuro-ectodermal, can cause multiple soft pigmented or non-pigmented skin tumors. Pigmentation often occurs late in the course of the disease. The tumors are usually situated along nerve trunks and in this way they may infiltrate any organ system. Subperiosteal or intraosseous nerve tumors produce devitalization of contiguous bone and cartilage by pressure. The generalized occurrence of such neurofibromas is often referred to as von Recklinghausen's neurofibromatosis. This condition is thought to have some familial distribution, has been reported in congenital form and is malignant in the degree to which it infiltrates vital structures. Mental deficiency is common in subjects whose lesions are life-sparing. All of the features of illness in this baby can be explained by a diagnosis of neurofibromatosis; that is my diagnosis.

Necropsy Findings: Dr. Bergstrom

The body was that of an emaciated white female, with a body length of 45 cm. and weight of 1,400 gm. The skin was icteric. A 2.5 cm. nodule was present in the left gluteal region. The brain showed the lack of development usually seen in a baby of the patient's age. The cranial nerves and meninges were not involved. The thymus weighed 1.5 gm. The mesenteric lymph nodes were enlarged, but the bronchial, mediastinal, and retroperitoneal lymph nodes were not. The peritoneal cavity contained a small amount of yellow fluid, but the pleural cavities were normal. The lungs grossly showed some emphysema and microscopically there was emphysema and atelectasis with mononuclear and polymorphonuclear cell infiltration of some of the alveolar walls. The heart was of normal size. Several firm nodules, varying from 1 to 4 mm. in diameter were present in the walls of the auricles and left ventricle. The liver was slightly enlarged and the cut tissue was bile stained. Microscopically the parenchymal cells showed fatty metamorphosis and were bile stained, with bile thrombi plugging many of the bile capillaries. The stomach mucosa was covered with mucus and what appeared to be old blood. The entire intestinal tract showed many thick umbilicated areas on the mucosal surface, measuring from 5 to 10 mm. in

diameter and 2 to 4 mm. in thickness; some were quite firm and apparently calcified. These tumors were most numerous in the wall of the colon and resembled Peyer's patches in appearance. Microscopically, the surface of the bowel showed a loss of surface epithelium in some places. In the tumor bearing areas the submucosal portion had a thickness up to ten times the normal due to the presence of neurofibromatous neoplasms. The bones of the skull and the long bones above and below the knees showed involvement with tumors similar to those noted in other part of the body. Nodules were present along the femurs and longitudinal section of these bones disclosed cyst-like masses with a yellow periphery and a gelatinous center. The cartilage and metaphyses were not involved. Nodular tumors were found along the course of several nerves retroperitoneally on the posterior abdominal wall.

The nodules which extensively involved the various portions of the body as described had a uniform microscopic appearance. The centers were nearly avascular and were made up of a collagenous material which in some tumors was undergoing degeneration; polymorphonuclear leukocytes were present. Toward the periphery of these rounded tumors there was an increase in cellularity, the tissue being a combination of connective tissue and nerve fibers, the outermost layers being spindle and stellate shaped with numerous blood vessels. These tumors are neurofibromas.

The clinical diagnosis and discussant's diagnosis:

Neurofibromatosis.

The pathological diagnosis:

1. Multiple neurofibromatosis involving:
   - Subcutaneous tissue
   - Skull and long bones
   - Myocardium
   - Intestinal wall
   - Retroperitoneal nerve trunks
2. Hemolytic icterus.
3. Fatty metamorphosis of liver.

Discussion: Dr. E. Clarence Rice

So far as I can remember, we have never had a patient at this hospital with such an extensive involvement of the various portions of the body with neurofibromas, as in the patient under the discussion. It is not too rare a disease in children, but to my knowledge it has never had a lethal effect in any of our other patients. We have one patient under observation who has a marked thoracic deformity due to the involvement of the chest wall. Heredity seems to play a part in von Recklinghausen's disease, it being inherited as a Mendelian dominant characteristic. No history of the disease in the relatives of this patient could be obtained. Like a number

of other tumors seen in young children, neurofibromas may lie dormant until puberty to assume an increase in size of the tumors at that time. Mental deficiency is sometimes observed. Pigmentation of the skin at the sites of these tumors is usually noted, but was not observed in this patient. Bone changes as seen in this patient are often found. Involvement of the cranial nerves may give rise to distressing intracranial symptoms. The microscopic appearance of these tumors is not that which the pathologist associates with malignant change. These tumors are benign tumors which have grossly caused destructive involvement of many of the important organs of the body producing essentially the same effects as a malignant tumor would. The only other malignant tumor of the nervous system with bone involvement which one might see in an infant of this age, would be a neuroblastoma.

EDITORIALS

EDITORIAL: THE ADOLESCENCE OF CLINICAL PROCEEDINGS

At the beginning of an eleventh year of publication, *Clinical Proceedings*, in keeping with the urges of adolescence, is making some changes. The front page is modified to include a picture of the new hospital buildings. The major portion of the "great remodeling" is now completed. Thus it is appropriate to remind readers monthly of these new and pleasant facilities and of the challenge they present the staff and friends of Children's Hospital to render proportionately greater service toward the fulfilment of the three main objectives of the institution as outlined in the first article of the constitution in 1870:

1. The medical and surgical treatment of sick children.
2. Instruction in the diseases of children.
3. Instruction of young women in the duties of nursing.

In recent years of course the spirit of these words has naturally extended to include as objectives not only the medical and surgical treatment of sick children but the guidance through health of well children and the psychiatric, social and rehabilitative aspects of medical and surgical care. All of these functions have been advanced by the inclusion of clinical research programs. Those close to *Clinical Proceedings* feel that this publication has become so linked with the fulfilment of these objectives that its future depends upon the spirit in which the staff and friends of Children's accept the challenge of the new hospital facilities, and that its content is a barometer of the hospital's health.

Doctor Rice

Another change in *Clinical Proceedings* is the resignation of Dr. E. Clarence Rice as Editor-in-Chief; fortunately Dr. Rice will continue as a Managing Editor. Dr. Rice has been an Editor and a guiding hand of the *Proceedings* since Volume I, Number 1, just as he has been in his quiet way a source of information and stimulation to the residents and staff of this hospital. For his steady and wise hand through infancy and childhood, *Clinical Proceedings* says "Thanks". It is reassuring to have him as a right hand "consulting pediatrician" through adolescence.

EDITORIAL: ADEQUACY AND POTENCY OF PERTUSSIS VACCINE

In this issue, Dr. Seebode reviews information specifically on the value versus the detriment of pertussis immunization for convulsive or potentially convulsive children. He deals more generally with the subject of the toxicity of pertussis vaccine. In the text, there are two statements which warrant further thought.

> The first is: "the fact that more children are being immunized all the time does reduce the risk of a non-vaccinated child developing pertussis."

The implication in this statement is that the immunized individual does not harbor *Hemophilus pertussis* nor develop symptoms from it. Neither of these implications is necessarily based in fact. Even the most optimistic reports on pertussis vaccine recognize that it is the least effective of the commonly used antigens for protection against clinical disease, the attack rate being as high as 35 per cent among immunized individuals. Recent work would indicate further that children apparently well protected by active immunization against *clinical* whooping cough, do indeed become infected with *H. pertussis* and show signs of mild bronchitic respiratory disease. They are presumably then silent carriers of infection. If these points are true, then it is not so tenable to argue against pertussis immunization of a select group such as convulsive children merely on the premise that nearly all their contacts are immunized.

The review paper also mentions the need for better standardization of pertussis vaccines. Actually recent efforts toward this end have been most encouraging:

1. The gross inadequacy of using quantity of bacteria as a measure of human protective ability has been recognized, there often being a fourfold difference in protection afforded by vaccines containing the same number of bacteria.

2. As a result of this knowledge, the National Institutes of Health directed on August 1, 1953 that potency requirements be based on the value of the total human immunizing dose instead of the number of bacteria in the vaccine. An arbitrary value of 12 units was assigned to the amount of vaccine equivalent to a total immunizing dose of reference vaccine (in which 96,000 million bacteria are adequate to afford reasonable protection for a child). This amount is measured in the laboratory by mouse protective ability of vaccine; hence the term 12 mouse protective units is often used. Each lot of pertussis vaccine must currently contain between 8 and 36 units in controlled tests to be accepted as providing a total human immunizing dose in a specified volume. It is true that there is still variation in potency but it is far less marked than it was. In time it can be expected that a narrower range of limits will be obtained.

This new regulation has the obvious advantage of assuring the adequacy of vaccine. A less obvious but equally important advantage is that there are also imposed *upper limits* of potency. Formerly up to 33 per cent of pertussis vaccines contained greater than the prescribed potency per total dose. If toxicity is in any way related to potency, it is encouraging to know that it "should now be possible to avoid the use of excessive dosage and thereby probably reduce the incidence of reactions."[1] If this is true, here is another factor that would diminish the risk of immunizing all children including those who are known to convulse.

REFERENCE

1. Pittman, M., Variability in the Potency of Pertussis Vaccine in Relation to the Number of Bacteria, J. Pediat., **45**: 57, 1954.

NEWS AND NOTES

EXCERPTS FROM THE PROGRAM AT THE DEDICATION OF THE DOCTOR JOSEPH STILES WALL MEMORIAL AUDITORIUM ON NOVEMBER 18, 1954

Invocation: Doctor Malcolm Marshall
Rector, St. Margaret's Episcopal Church

Almighty God, we remember this day before thee thy faithful servant, Joseph, and we pray thee that, having opened to him the gates of larger life, thou wilt receive him more and more into thy joyful service; . . . and, healing all, continue, we beseech thee, this gracious work among us. Cheer, heal and sanctify the sick; grant to the physicians, surgeons and nurses wisdom and skill, tenderness and patience and assist with thy blessing all

who are seeking to prevent suffering and to forward thy purposes of love; through Jesus Christ our Lord. Amen.

Presiding: JUDGE J. EDGAR MURDOCK
President, Board of Directors

The Medical Staff of Children's Hospital of the District of Columbia informed the Board of Directors, when the latter was considering the construction of this new hospital, that it needed an auditorium. Today we are here to dedicate such an auditorium in memory of Dr. Joseph Stiles Wall who, as Chief of Staff, presented the request to the Board of Directors. .... Dr. Wall kept plugging, on behalf of the Staff, for an auditorium and . . . a great many people participated to make the auditorium a reality. The Board of Directors thanks all those who have made this possible.

The only purpose of this hospital is to provide the best medical care possible for the little patients who are brought here. . . . That is done largely through the skill of our Staff. The rest of us are merely trying to provide the Staff with the best equipment and service obtainable so that the physicians on the Staff may give superb care and continue to make this hospital an asset to this community. Dr. Wall has been and will continue to be a great inspiration for all of us and particularly for the Medical Staff.

Speaking for the Board of Directors, many of whom have served this hospital longer than I have, I say to the Staff: Here is your auditorium. We are delighted to provide it for your use and we are confident that you will carry on, as you have in the past, to merit the approval of Dr. Joseph Stiles Wall.

Acceptance: DOCTOR EDGAR P. COPELAND

It is my very pleasant duty this noon to express the very sincere appreciation of the Medical Staff for this very wonderful addition to the teaching facilities of Children's Hospital and to the care of the patients. . . .

It was not anticipated that Doctor Joseph Stiles Wall would be absent on this occasion. It is wonderful to know that Doctor Wall knew that this auditorium was to be available and it must have been very pleasing to him to know that it would be named after him, in recognition of the devoted work he put into this facility.

I like to feel that, while Doctor Wall is not here with us today, his great spirit is; and I would like to hold the fantasy that he is looking down here and being with us, enjoying the realization of his fondest dream.

Speaker: DOCTOR HARRY BAKWIN
President-elect, American Academy of Pediatrics

The Academy of Pediatrics is deeply honored at this opportunity to pay tribute to one of its most distinguished members.

Doctor Joseph Stiles Wall was one of the small group of energetic, far-sighted visionaries who founded the Academy in 1931. From its inception, almost up to the time of his death, Dr. Wall served the Academy without interruption.

During the troubled war years he worked with various government agencies in the best interests of the welfare of children and in 1946 he received the only citation ever given by the Executive Board for service above and beyond what could be expected of him in the normal performance of duty. The children of America—indeed the children of the world—could have no more capable or effective champion. His analysis of the E.M.I.C. plan in 1944 was a masterpiece of clear thinking, intimate acquaintance with the data and concise writing.

As Chairman of the Legislative Committee for many years, Dr. Wall was tireless in following national legislation relating to child health as it developed in the halls of Congress. Time and time again, at great personal sacrifice, he appeared before committee hearings and presented the Academy's views on proposed legislation. He was further responsible for the preparation of the many changes in the Constitution and By-Laws, made necessary by the rapid growth of the Academy and which culminated in the reorganization in 1949–50.

In 1944–45 Dr. Wall was President of the Academy.

These were the official associations of Dr. Wall with the Academy. Of equal importance were his sterling personal qualities—a friendliness, a graciousness, a finesse of character—which made him stand out and brought him the friendship and respect of everyone of the Society. We honor him, not only for his outstanding contribution to the work and development of the Academy of Pediatrics but as a man who unselfishly and untiringly gave of himself for the betterment of medicine and American children.

Speaker: Doctor Wallace M. Yater
Past-President, Medical Society of District of Columbia

My remarks will be directed more to those of you who did not have the privilege of direct contact with Dr. Wall's warm personality.

I am happy to say that Dr. Wall was one of my dearest friends over a long period of years. Besides our close friendship, the memory of which I cherish, I was privileged to know Dr. Wall in five different ways.

First: Dr. Wall was my professor of pediatrics 35 years ago. I remember him as a lucid, interesting and entertaining lecturer. He had the rare gift of placing himself on the level of the student, which is the essence of the perfect teacher. Dr. Wall was on the faculty of Georgetown University for 47 years and many hundreds of students had the benefit of his instruction.

Second: I was associated with Dr. Wall on the Executive Faculty of Georgetown University School of Medicine. I am happy to say that we usually saw things "eye to eye." Dr. Wall gave me great encouragement over the period of 16 years that I was head of the Department of Medicine. He never committed himself without the most careful consideration and his judgment, in a ticklish problem, was always accepted by the faculty.

Third: I knew Dr. Wall as a co-worker in the Medical Society. Among other accomplishments, he brought about enactment of the law to regulate the Practice of the Healing Arts in the District of Columbia. For this important piece of work he received a scroll from the Society, in 1929, which read "for outstanding services to the medical profession of Washington," and for "contributions in the field of pediatrics and . . . unselfish devotion to the health needs of the children of the District of Columbia." In 1948 Dr. Wall was given the award for meritorious service by the Society and in 1951 his friends had a portrait painted of him and presented to the Society, where it hangs in the board room.

Fourth: I knew Dr. Wall as pediatrician to all four of my children. They all loved him and their mother was always comforted by his visit. He always left a trinket for the little patient.

Fifth: I knew Dr. Wall in the capacity of his personal physician. Fortunately, he was in good health until the age of 70. As a patient he was stoical, uncomplaining and co-operative. The only fault I could find was that he was so unconcerned about himself that it was difficult to get him in for periodic checkups. In his final illness he was totally unafraid.

I shall close by quoting from Mr. Theodore Wiprud's obituary of Dr. Wall in the Medical Annals of the District of Columbia. I cannot express as well the sentiments of all who knew him.

> "To those who knew Dr. Wall best, he was kindly, just and loyal. He had an infectious smile and a good word for everyone. That he had plenty of courage he demonstrated when occasion required but even then his gentle manner turned away the wrath of those who did not agree with him. His poise and fairness were particularly evident when presiding at a meeting, which he did with consummate skill. Never wordy and invariably impartial, he always had the situation well in hand.
>
> Dr. Wall was an unusually gifted speaker. What he had to say he said with disarming charm, yet his words had the force of conviction which left no doubt in his hearers' minds where he stood. . . . He was the kind of man that one felt better for having talked to."

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